

- 3 Murakami S, Mizobuchi M, Nakashiro Y, Doi T, Hato N, Yanagihara N. Bell's palsy and herpes simplex virus: identification of viral DNA in endoneurial fluid and muscle. *Ann Intern Med* 1996;124:27-30.
- 4 Adour KK. Current concepts in neurology: diagnosis and management of facial paralysis. *N Engl J Med* 82;307:348-51.
- 5 House JW, Brackmann DE. Facial nerve grading system. *Otolaryngol Head Neck Surg* 1985;93:146-7.
- 6 Dresner SC. Ophthalmic management of facial nerve paralysis. *Focal points*. San Francisco: American Academy of Ophthalmology, Jan 2000.
- 7 Stanek G, Strle F. Lyme borreliosis. *Lancet* 2003;362:1639-47.
- 8 Dobie RA. Tests of facial nerve function. In: Cummings CW et al, eds. *Otolaryngology head and neck surgery*. New York: Mosby, 1998:2757-66.
- 9 Sweeney CJ, Gilden DH. Ramsay Hunt syndrome. *J Neurol Neurosurg Psychiatr* 2001;71:149.
- 10 Grogan PM, Gronseth GS. Practice parameter: steroids, acyclovir, and surgery for Bell's palsy (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology* 2001;56:830-6.
- 11 Ramsey MJ, DerSimonian R, Holt MR, Burgess LP. Corticosteroid treatment for idiopathic facial nerve paralysis: a meta-analysis. *Laryngoscope* 2000;110:335-41.
- 12 Williamson IG, Whelan TR. The clinical problem of Bell's palsy: is treatment with steroids effective? *Br J Gen Pract* 1996;46:743-7.
- 13 Shafshak TS, Essa AY, Bakey FA. The possible contributing factors for the success of steroid therapy in Bell's palsy: a clinical and electrophysiological study. *J Laryngol Otol* 1994;108:940-3.
- 14 Hato N, Matsumoto S, Kikaki H, Takahashi H, Wakasaka H, Honda N, et al. Efficacy of early treatment of Bell's palsy with oral acyclovir and prednisolone. *Otol Neurotol* 2003;24:948-51.
- 15 Lagalla G, Logullo F, Di Bella P, Provinciali L, Ceravolo MG. Influence of early high-dose steroid treatment on Bell's palsy evolution. *Neurol Sci* 2002;23:107-12.
- 16 Salinas RA, Alvarez G, Alvarez MI, Ferreira J. Corticosteroids for Bell's palsy (idiopathic facial paralysis). *Cochrane Database Syst Rev* 2002;(1): CD001942.
- 17 Burgess LP, Yim DW, Lepore ML. Bell's palsy: the steroid controversy revisited. *Laryngoscope* 1984;94:1472-6.
- 18 De Miranda P, Blum MR. Pharmacokinetics of acyclovir after intravenous and oral administration. *J Antimicrob Chemother* 1983;12(suppl B):29-37.
- 19 Snoeck R, Andrei G, De Clercq E. Current pharmacological approaches to the therapy of varicella zoster virus infections: a guide to treatment. *Drugs* 1999;57:187-206.
- 20 De Diego JL, Prim MP, De Sarria MJ, Madero R, Gavilan J. Idiopathic facial paralysis: a randomized, prospective, and controlled study using single-dose prednisone versus acyclovir three times daily. *Laryngoscope* 1998;108:573-5.
- 21 Sipe J, Dunn L. Aciclovir for Bell's palsy (idiopathic facial paralysis). *Cochrane Database Syst Rev* 2001;(4):CD001869.
- 22 Axelsson S, Lindberg S, Stjernquist-Desatnik A. Outcome of treatment with valacyclovir and prednisone in patients with Bell's palsy. *Ann Otol Rhinol Laryngol* 2003;112:197.
- 23 Murakami S, Hato N, Horiuchi J, Honda N, Gyo K, Yanagihara N. Treatment of Ramsay Hunt syndrome with acyclovir-prednisone: significance of early diagnosis and treatment. *Ann Neurol* 1997;41:353-7.
- 24 Salman MS, MacGregor DL. Should children with Bell's palsy be treated with corticosteroids? A systematic review. *J Child Neurol* 2001;16:565-8.
- 25 Fisch U. Surgery for Bell's palsy. *Arch Otolaryngol* 1981;107:1-11.
- 26 Beurskens CH, Heymans PG. Positive effects of mime therapy on sequelae of facial paralysis: stiffness, lip mobility, and social and physical aspects of facial disability. *Otol Neurol* 2003;24:677-81.

(Accepted 8 June 2004)

## Lesson of the week

# Rare causes of haemoptysis in suspected pulmonary embolism

M S Warburton, M A Jackson, R Norton, M Bhabra

When a patient presents with haemoptysis and pleuritic chest pain, a pulmonary embolism is an important and common diagnosis to consider. There is a tendency in busy medical admissions units to start treatment of certain conditions without thorough investigation, with the intention of reducing delays in starting treatment. Patients are usually treated for suspected pulmonary embolism with heparin early to reduce mortality and morbidity. It is important, however, to remember other less common causes of haemoptysis.

## Case report

A 59 year old woman was admitted to our medical assessment unit with chest pain and haemoptysis. She had experienced pleuritic left sided, chest wall pain intermittently for the previous week, with gradually increasing intensity. On the day of admission she had also produced about a cupful of bright red blood while coughing. She had no medical history of note, except that she was a smoker.

On admission the patient was in distress but not objectively dyspnoeic—her respiratory rate was not raised and her oxygen saturations on air were 97%. She did not show any signs of shock; she had no tachycardia and her systolic blood pressure remained around 120 mm Hg throughout. Chest examination showed some decreased air entry at her left lung base with a loud pleural rub. She had no cardiac murmurs, and no clinical evidence was found of a deep venous thrombosis in her legs or pelvis.

The admitting doctor's differential diagnosis included pulmonary embolism, pneumonia, and carcinoma of the lung; a plain chest radiograph and routine blood tests were ordered including a D-dimer assay.

The chest x ray film (fig 1) showed moderate left basal shadowing with blunting of the right costophrenic angle. There was no cardiomegaly, and, importantly, the mediastinum appeared normal.

Blood tests indicated a mild anaemia (haemoglobin 96 g/l), a raised white cell count (28.5, neutrophils 25.9)

**Do not automatically treat a suspected pulmonary embolism with heparin; consider other diagnoses first**



**Fig 1** Chest x ray film showing moderate left basal shadowing with blunting of the right costophrenic angle

Department of General Medicine, New Cross Hospital, Wolverhampton WV6 7OQ  
M S Warburton senior house officer  
M A Jackson consultant physician in general, renal, and metabolic medicine

Walsgrave Hospital  
R Norton consultant cardiothoracic surgeon  
M Bhabra consultant cardiothoracic surgeon

Correspondence to: M Warburton matthew\_karen@sayang.freemove.co.uk

BMJ 2004;329:557-8



**Fig 2** Spiral computed tomogram showing aneurysm of lower part of descending thoracic aorta with surrounding haematoma

and a normal prothrombin time. A D-dimer assay was greatly raised at 3893 (normal <250) ng/ml. Inflammatory markers were raised, with an erythrocyte sedimentation rate of 73 seconds and a C reactive protein of 411 mg/l. Her alkaline phosphatase was mildly raised, though corrected calcium and the rest of her liver enzymes were normal. Renal function was normal and blood cultures were subsequently reported as “no growth.”

Based on the chest radiograph and inflammatory markers, treatment was initially begun for a left basal pneumonia with oral co-amoxiclav (amoxicillin 500 mg, clavulanic acid 125 mg). She was not started on low molecular weight heparin as is the case normally for suspected pulmonary embolism. She was admitted overnight and seen in the morning by the on-call consultant. She had made no improvement and needed opioids to control her pain.

The consultant on call was concerned that this was an atypical presentation for a pulmonary embolus and arranged urgent spiral computed tomography of the patient's thorax to elucidate the cause of the chest pain. The tomogram showed an aneurysm of the lower part of the descending thoracic aorta with surrounding haematoma (fig 2). She was immediately transferred to the local cardiothoracic unit for surgery.

Through a posterolateral thoracotomy and with partial cardiopulmonary bypass, the aneurysmal segment of the aorta was replaced with a Dacron tube graft. There were no postoperative complications. A mixed growth of Gram positive and Gram negative

cocci was cultured from the contents of the aneurysm, and so although the patient was clinically well, she was treated with a course of intravenous antibiotics.

## Discussion

Thoracic aneurysms are rare, estimated at 6 per 100 000 a year.<sup>1</sup> In contrast, pulmonary embolism is more common (60 to 70 cases per 100 000 a year).<sup>2</sup> We know that “common things occur commonly,” and since pulmonary emboli have a considerable mortality associated with them, there is a great temptation to treat before the diagnosis is confirmed. The British Thoracic Society recommends initial investigation with D-dimer assays and, if combined with a high clinical suspicion, treatment with low molecular weight heparins until imaging is available. In this case, however, to have given any kind of anticoagulation might have proved fatal.

The society's guidelines also state that if a pulmonary embolus is ruled out (or thought unlikely) then high resolution or multislice computed tomography is necessary to identify the true nature of the pain.

The clues that pointed this case towards an “atypical” chest pain were the high white cell count; the large level of haemoptysis; no significant hypoxia; no evidence of deep venous thrombosis on clinical examination; and a pain much more severe than would be expected for pulmonary embolism.

This mode of presentation is also somewhat unusual for a thoracic aortic aneurysm in terms of the nature of the pain and the presence of haemoptysis. The location of the aneurysm was such that it was not apparent on the plain chest radiograph. The identification of micro-organisms would suggest an infective aetiology.

We advise to always consider alternative diagnoses of pulmonary embolism if atypical features are present.

MB now works at New Cross Hospital, Wolverhampton.

Contributors: All four authors were involved in the treatment of the patient in the case study, and all were involved in writing the paper. MAJ is the guarantor.

Funding: None.

Competing interests: None declared.

1 Bickerstaff LK, Pairolero PC, Hollier LH, Melton LJ, van Peenen HJ, Cherry KJ, et al. Thoracic aortic aneurysms: a population-based study. *Surgery* 1982;92:1103-8.

2 British Thoracic Society Standards of Care Committee Pulmonary Embolism Guideline Development Group. British Thoracic Society guidelines for the management of suspected acute pulmonary embolism. *Thorax* 2003;58:470-83.

*bmjlearning.com*

## Occupational asthma

Occupational asthma is the most common form of occupational lung disease in developed countries. As more new chemicals are produced, people will become exposed to more respiratory sensitisers and irritants. About 10% of patients with adult onset asthma have occupational asthma, and it can affect not only their health and quality of life but also their jobs and finances. For these reasons, you should always consider the diagnosis in people who develop asthma as adults. Since the prognosis is related to the duration of the exposure, it is important to diagnose and treat patients promptly.

Many myths about occupational asthma persist despite being disproved by evidence. For example, not all patients feel better

when they take a weekend off work: many patients require 7-10 days for symptoms to resolve. And you don't need to tell patients to stay away from work while the diagnosis is being considered unless they are very ill.

To find out more myths and facts on occupational asthma, try BMJ Learning's new module on *bmjlearning.com*. Our interactive learning website offers online resources to train and test your skills in a variety of clinical and non-clinical topics.

Kieran Walsh *editorial registrar, BMJ Learning*  
(*bmjlearning@bmjgroup.com*)